

E-Pearl: Brachial artery embolus mimicking acute stroke

Christine Holmstedt, DO
Marc Chimowitz,
MBChB

Address correspondence and
reprint requests to Dr. Christine
Holmstedt, Harborview Office
Tower, 19 Hagwood Ave., MSC
805, Medical University of South
Carolina, Charleston, SC 29464
holmstedt@musc.edu

PEARL

- Limb arterial embolism should be considered in the differential diagnosis of acute monoparesis because the diagnosis may be missed if the other typical manifestations of this presentation (pain, pallor, pulselessness, sensory loss, and coolness of the arm) are overlooked. Although the rapid identification of acute ischemic stroke is essential to the timely delivery of thrombolytics, care must be taken to obtain the relevant history and to ensure that important signs are not missed whether the evaluation of the patient is done at the bedside or by telemedicine.
- Rapid and accurate diagnosis of ischemic stroke is essential for timely and appropriate treatment with thrombolytic therapy. Occasionally, patients who present with acute stroke mimics (i.e., disorders that resemble stroke such as seizure, hypoglycemia, and psychogenic disorder) are incorrectly treated with thrombolytic therapy. The incidence of stroke mimics varies greatly and is dependent on many variables including time of evaluation and experience of first responder.¹ To our knowledge, the literature pertaining to stroke mimics or misdiagnosis does not include limb ischemia due to extremity embolism.^{2,3} We present a case of a patient with acute right upper extremity weakness, who was referred through a stroke telemedicine evaluation system from a distant emergency department. History and examination by telemedicine led to the diagnosis of a right brachial artery embolus rather than a stroke, which resulted in emergency surgical embolectomy rather than IV thrombolytic therapy.

CASE REPORT An 85-year-old woman presented to an emergency department with acute onset of right upper extremity pain and weakness. She denied sitting with her arm propped up or falling asleep in an unusual position. Her medical history included

paroxysmal atrial fibrillation, hypertension, dyslipidemia, congestive heart failure, and coronary artery disease. The patient was not anticoagulated because of a recent gastrointestinal bleeding episode.

The emergency department physician diagnosed acute stroke and requested a telemedicine consultation. The patient's blood pressure was 160/70 mm Hg, and a rhythm strip demonstrated normal sinus rhythm at 80 beats per minute. On neurologic examination, the right upper extremity strength revealed no effort against gravity with some preserved strength in wrist and finger extension. The patient could not localize touch on the right arm. Findings from the remainder of the neurologic examination, including speech and language, cranial nerves, coordination, and right lower extremity strength and sensation, were normal. Reflexes and plantar responses were not tested as part of the telemedicine stroke examination in this patient. A CT scan of the brain showed normal results.

Because pain was a prominent feature of the presentation, palpation of the right upper extremity for pulses was suggested to the emergency department physician. The right brachial and radial pulses were not palpable, and the right upper extremity was colder than the left upper extremity. An emergency right upper extremity ultrasound was performed, which revealed a right brachial artery occlusion. Intravenous heparin treatment was started, and the patient was transferred to the operating room for emergency right upper extremity exploration and embolectomy of the right brachial artery.

A transthoracic echocardiogram was negative for an intracardiac thrombus. The patient recovered and was discharged home 3 days later with aspirin therapy.

DISCUSSION This patient illustrates that brachial artery embolism should be considered in the differential diagnosis of paresis of the arm and that the diagnosis may be missed if the other typical manifestations of this presentation (pain, pallor, pulselessness, sensory loss, and coolness of the arm) are overlooked. The upper

extremity embolus was not suspected in this patient until the vascular neurologist obtained the history of pain, an uncommon presenting symptom of acute stroke. This additional information prompted a peripheral vascular examination and ultimately an upper extremity ultrasound.

Atrial fibrillation is thought to cause 80% of cases of arterial embolic occlusion.⁴ Upper extremity embolic occlusions represent approximately 23% of all peripheral limb emboli, with 61% occurring in the brachial artery.⁴ Symptoms of upper extremity arterial occlusion generally consist of pallor, pain, and paresthesias as well as a lack of brachial ulnar or radial pulses. Other manifestations including weakness, edema, distal limb rigidity, and anesthesia, which are believed to reflect secondary thrombosis or multiple emboli, are considered important prognostic factors.⁴ Historically, treatment of upper extremity embolism was conservative because the event was viewed as somewhat benign. However, in 1981, Haimovici⁴ described a series of 46 nonsurgically treated patients with upper extremity embolism of whom 31% developed gangrene, had a hand amputation, or died. In a review of 322 cases of arterial embolectomy from 6 reports, 9.3% of patients developed gangrene and 11.8% died.

Current treatment recommendations include immediate anticoagulation with heparin, surgical embolectomy, or intra-arterial thrombolytics and embolization. Randomized trials have suggested a benefit of thrombolytic therapy in the initial management of patients with peripheral arterial occlusion.^{5,6} Although limb salvage rates are similar between the thrombolytic and open embolectomy groups, the survival rate was improved in the patients randomized to the thrombolytic group.⁶ Regardless of management, prompt treatment is warranted because arm ischemia can lead to loss of arm or hand function.

With regard to the case presented, several possible outcome scenarios existed. A misdiagnosis of acute ischemic stroke may have led to treatment with IV

thrombolytic therapy, which may have been effective if the embolus was completely lysed. However, if the embolus had not lysed or was only partially lysed, this may have been interpreted as a failure of thrombolysis for acute stroke. In this patient, failure to recognize the brachial artery embolus could have resulted in further limb ischemia, gangrene, limb amputation, or death. Even if the brachial artery embolus had been recognized after unsuccessful thrombolysis, the use of recombinant tissue-type plasminogen activator may have necessitated delay of anticoagulation and embolectomy in an otherwise good surgical candidate.

Although the rapid identification of acute ischemic stroke is essential to the timely delivery of thrombolytics, care must be exercised to obtain a complete history and perform a rapid and thorough examination. Our case not only represents an unusual stroke mimic but also reiterates the importance of fundamental clinic skills in the evaluation of patients.

DISCLOSURE

Dr. Chimowitz has served on scientific advisory boards for Gore Corporation and Parexel; serves on the editorial board of *Neurosurgery*; has received research support from Boston Scientific, AstraZeneca, and the NIH/NINDS; and has served as an expert witness in medicolegal cases. Dr. Holmstedt reports no disclosures.

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Neurology[®]

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Neurology 2011;76:e86-e87

DOI 10.1212/WNL.0b013e3182190cc0

This information is current as of May 2, 2011

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